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Hypertrophy of the Heart

Electrocardiographic Distinction Between Physiologic and Pathologic Enlargement

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• *Electrocardiograms of marathon runners were examined to study hypertrophy of the heart due to prolonged physical exertion and to differentiate this from hypertrophy due to various disease states, especially essential hypertension, aortic valvular disease and coarctation of the aorta. The electrocardiogram of the marathon runners was characterized by a slow cardiac rate, high voltage of the QRS complexes and T waves in the standard and/or precordial leads with normal R/T ratios. There was moderate enlargement of the heart as observed on teleoroentgenogram. These findings are characteristic of physiologic hypertrophy of the heart and should be suspected among patients having a history of athletics calling for endurance. Immediately after running, all waves showed an increased voltage and the heart size decreased. The concept of the secondary T wave in hypertension as a part of the left ventricular strain pattern was challenged by the observation that the increased voltage of the R waves in lead V5 and other leads seen in marathon runners and in certain patients with hypertension, aortic stenosis, aortic insufficiency and coarctation of the aorta were not necessarily associated with typical discordant S-T segments and T waves. There was a higher incidence of dyspnea, angina pectoris and cardiac enlargement among hypertensive patients with discordant T waves than among hypertensive patients without these changes. Thus it is felt that the discordant waves are primary and are not merely secondary to the increased area of the R waves. Primary T waves suggest myocardial disease, possibly anoxia of the subendocardium.*

THE PRIMARY PURPOSE of this study is to describe the electrocardiographic features of physiologic hypertrophy of the heart due to prolonged physical exertion, and to differentiate that condition from pathologic hypertrophy due to various disease states, especially essential hypertension. The study was undertaken as the result of the observation that marathon runners at rest showed cardiac enlargement on teleoroentgenograms and high voltage of the QRS complexes and T waves in electrocardiograms. An additional purpose is to evaluate the concept of the secondary T wave which is considered a part of the electrocardiographic pattern of left ventricular strain. This concept suggests that increased left ventricular pressure results in high voltage of QRS complexes with increased areas under

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TABLE 1.—*Amplitudes of the highest R waves (in millimeters) in the six precordial leads in 40 non-runners and 40 runners*

	Non-runners	Runners: Before Running	Runners: After Running
Mean	18.6	24.1	25.8
Min.	8.0	8.0	4.0
Max.	31.0	52.0	42.0
S.D.±	5.1	8.0	7.0
C.V.± %	26.0	33.0	27.0

S.D. = Standard deviation. C.V. = Coefficient of variation.

TABLE 2.—*Amplitudes of the deepest S waves (in millimeters) in the six precordial leads in 40 non-runners and 40 runners*

	Non-runners	Runners: Before Running	Runners: After Running
Mean	12.7	19.8	21.7
Min.	0.0	4.0	2.0
Max.	29.0	38.0	45.0
S.D.±	5.3	4.5	8.0
C.V.± %	41.0	23.0	36.0

TABLE 3.—*Amplitudes of the tallest T waves (in millimeters) in the six precordial leads in 40 non-runners and 40 runners*

	Non-runners	Runners: Before Running	Runners: After Running
Mean	7.7	9.2	12.6
Min.	2.5	2.0	3.0
Max.	14.0	17.0	22.0
S.D.±	2.8	3.6	5.1
C.V.± %	36.0	38.0	40.0

the waves and, as a consequence, the occurrence of S-T segments and T waves which are discordant with the major deflection of the QRS complexes. These waves are considered secondary to the large waves of the QRS complexes and do not, therefore, in themselves indicate myocardial disease. This relationship is illustrated by the ventricular ectopic beat.

METHODS AND MATERIALS

The study was carried out on 165 marathon runners, most of whom had been in training for 26-mile races for over five years. In six instances, teleoroentgenograms of the chest were taken before and immediately after running to determine cardiac size. The electrocardiograms and teleoroentgenograms of 40 patients with essential hypertension, of ten patients with coarctation of the aorta and of one patient with rheumatic aortic stenosis and regurgitation were studied. Also electrocardiograms and teleoroentgenograms of 40 normal men with height, weight and age similar to the runners were employed for controls. Cardiac areas were determined (as by Hodges and Eyster[2]) from the teleoroentgenograms with the formula: $A = \frac{\pi}{4} L B$.* Electrocardiograms

*A = cardiac area in square centimeters; L = long diameter of heart in cm.; B = broad diameter in cm.

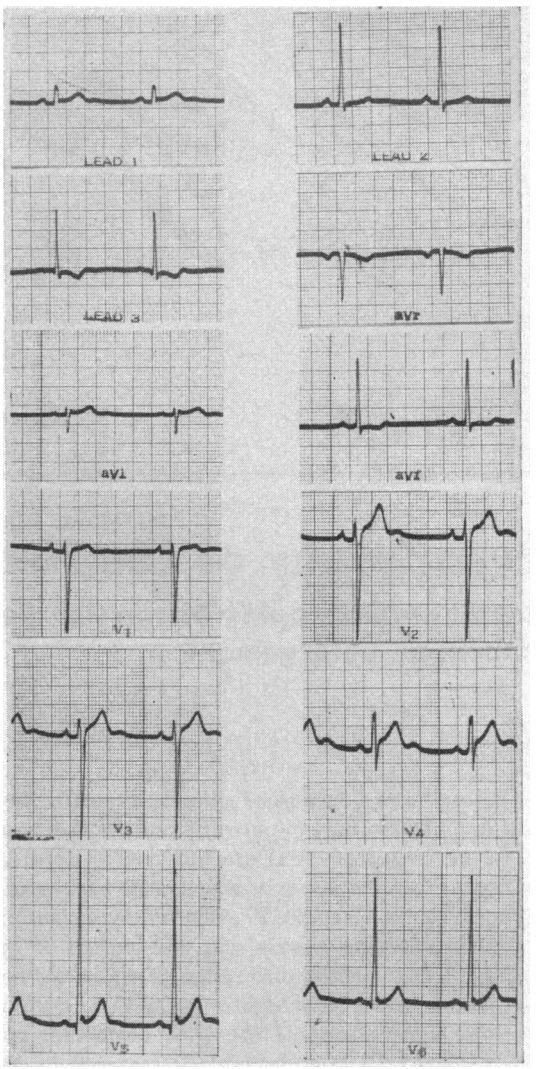

Figure 1.—Electrocardiogram of a runner at rest showing high voltage of the QRS complexes in lead V5 and other leads.

were standardized so that 1 millivolt produced a string or stylus deflection of 10 millimeters. Tracings were taken on Cambridge string and Sanborn photographic and direct writing electrocardiographic instruments. The areas under certain waves of the electrocardiogram were measured by counting the squares enclosed by the waves, with an accuracy of 15 per cent, and are reported in microvolt seconds (each small square on the electrocardiogram has an area of 4.0 microvolt seconds).

RESULTS

The effect of running on the electrocardiograms of 40 runners was determined by taking tracings before and immediately after they ran 26 miles, and comparison was made with the tracings of 40 control non-runners of similar age, height and weight.

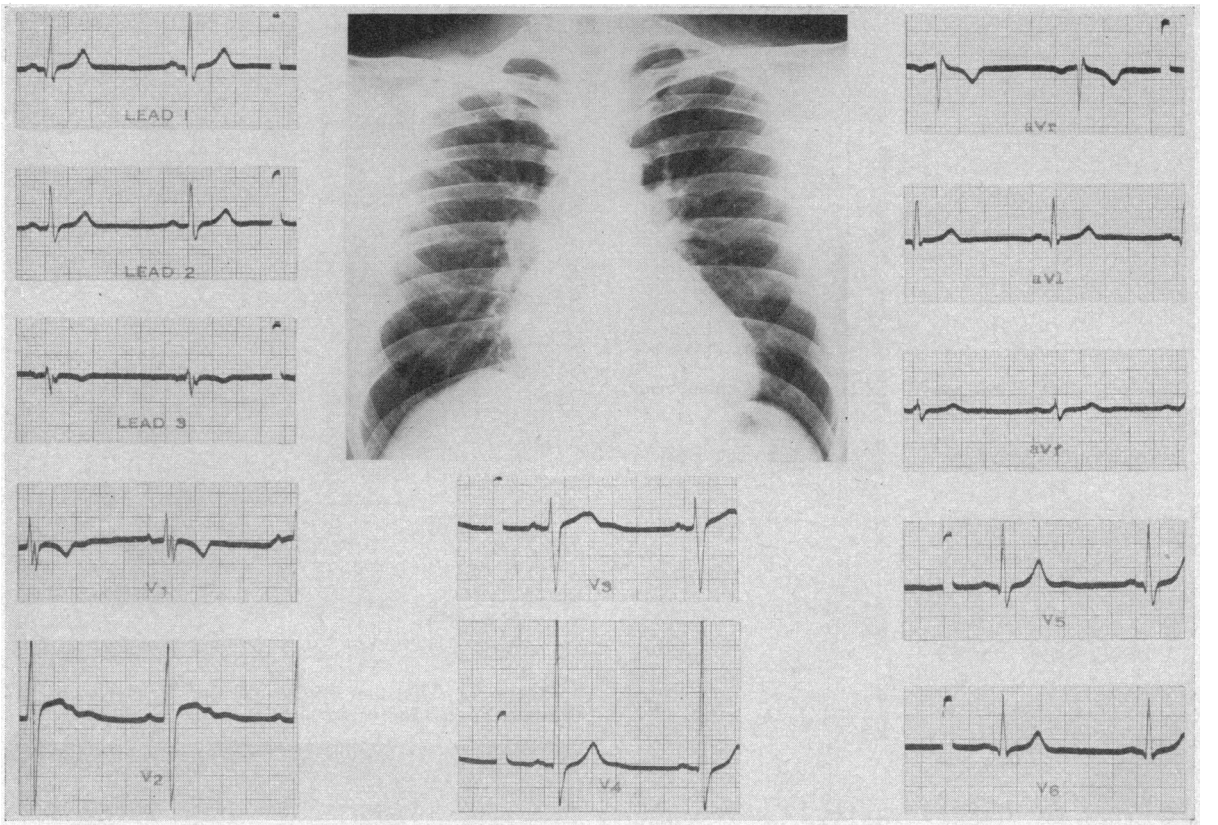

Figure 2.—Electrocardiogram of a runner at rest with R wave amplitudes in lead V5 which are at the upper limit of normal. The areas under the R waves and T waves in this lead are greater than the average for normals. The teleoroentgenogram shows the presence of cardiac enlargement.

The cardiac rate of the runners at rest averaged 56 beats per minute (range 42 to 76) and of the non-runners 72 (range 58 to 85).

The amplitudes of the highest R waves in the six precordial leads are shown in Table 1. The average was 18.6 mm. in the non-runners, and 24.1 mm. in the runners at rest, while after running it was 25.8 mm. Ten per cent of the tracings of the runners at rest exceeded the upper limit of normal for the non-runners, which is 31.0 mm.

The amplitudes of the deepest S waves in the six precordial leads, usually lead V2, were measured (Table 2). The average for the non-runners was 12.7 mm. as compared with 19.8 mm. for the runners at rest; after running the average value was 21.7 mm. Twelve per cent of the runners had S waves that exceeded the upper limit of normal for the non-runners, which was 29.0 mm.

The amplitudes of the tallest T waves in the six precordial leads are shown in Table 3. The average for the non-runners was 7.7 mm. compared with the runners at rest of 9.2 mm., while after running it was 12.6 mm. Sixteen per cent of the runners had T waves which exceeded the upper limit of normal for non-runners in this series, which was 14.0 mm.

Typical electrocardiograms of runners taken at rest and after running are illustrated in Figures 1 to 5. High voltage of the QRS complexes, especially in lead V5, is shown in Figure 1. The R waves measure 46.0 mm. in this lead. The upper limit of normal in a recently published series was 17.0 mm.[5] In the control group of 40 subjects in the present study the upper limit of normal was 31.0 mm. (Table 1).[1] In the patient whose tracing is shown in Figure 1, the areas under the R waves and T waves in lead V5 were greater than the average for the normal subject (*vide infra*). The sum of the amplitudes of the R waves in lead V5 added to the depth of the S waves in lead V1 was 68.0 mm., which exceeds the upper limit of normal of 35.0 mm.[5] and suggested the presence of left ventricular enlargement.

The amplitude of the R waves in lead V4 of the electrocardiogram shown in Figure 2 are at the upper limit of normal. The areas under the R waves and T waves are greater than the average for the normals (*vide infra*). The teleoroentgenogram shows the presence of cardiac enlargement.

R-R prime waves which were encountered in three of the runners are shown in Figure 3. The QRS complexes are not widened. This suggests strain on the

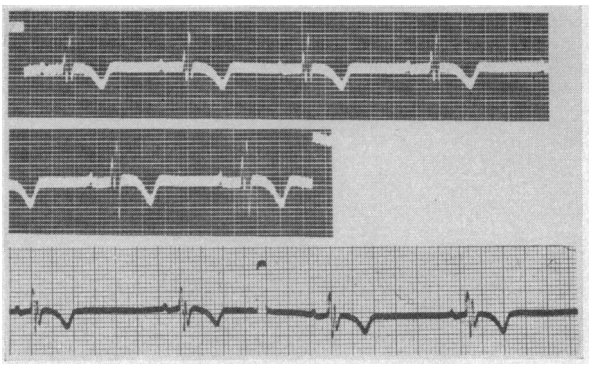

Figure 3—R-R prime waves in lead V1 in three runners at rest.

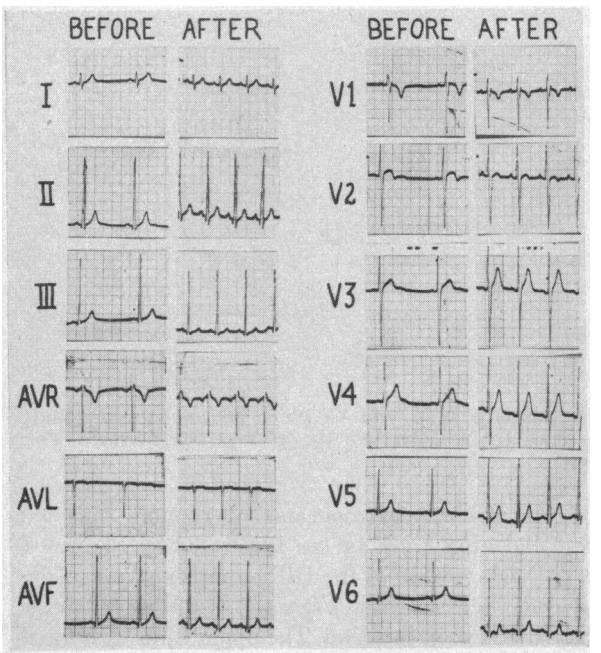

Figure 4.—High voltage of the QRS complexes in lead aVf before and after running 26 miles. Immediately after running there is an increase of the voltage of the QRS complexes especially in leads V4, 5 and 6.

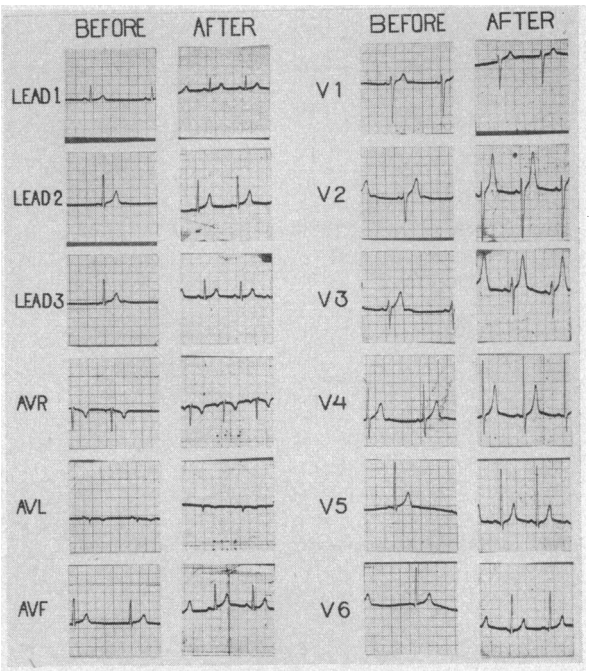

Figure 5.—The amplitudes of the T waves in V2 through V5 are increased after running 26 miles.

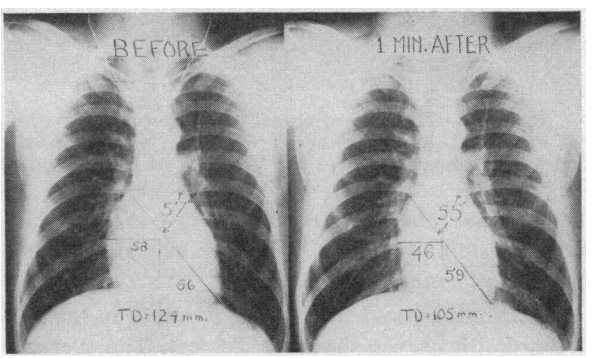

Figure 6.—A decrease in the size of the cardiac shadow occurs immediately after running 26 miles. There is no detectable change in the amount of air space between the heart and the chest cage after running.

right ventricle although incomplete right bundle branch block cannot be ruled out with certainty.

High voltage of the QRS complexes in lead aVf in a marathon runner before and after running 26 miles is shown in Figure 4. These waves are 37.0 mm. tall and exceed the upper limit of normal of 20.0 mm.[5] Likewise there is high voltage of the QRS complexes in lead 2 as shown by high R waves which are 37.0 mm. The amplitudes of these waves exceed the upper limit of normal of 22.0 mm.[5] Immediately after running an increased voltage of the QRS complexes in leads V4, 5 and 6 is seen (Figure 4).

Tall T waves are common in most of the chest leads in runners at rest. There is an absolute increase in the amplitude of the T waves after running, as the sum of all the T voltages in the 12 leads after running is greater than that before running (Figure 5).

Immediately after running there is a decrease in cardiac size with no significant change in the amount of air space between the heart and chest cage (Figure 6). The transverse diameter of the heart before running was 124.0 mm. as compared with 105.0 mm. immediately after running. Similar changes were seen in five other subjects in whom study of this type was made.

Measurements of the transverse, broad and long diameters of the heart, cardiac areas and great vessels diameters were analyzed (Table 4). All cardiac measurements were increased in the runners at rest, as compared with the normal control group. The

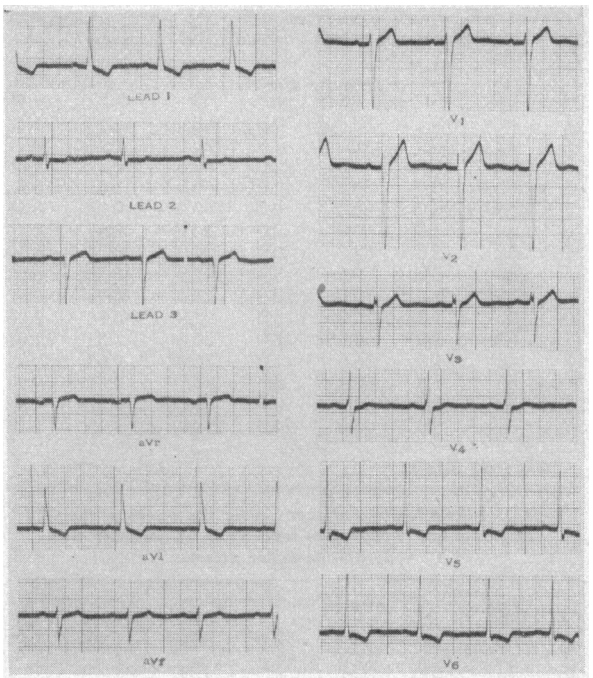

Figure 7.—Electrocardiogram showing typical "left ventricular strain pattern." The T waves and S-T segments are discordant with the main deflection of the QRS complexes.

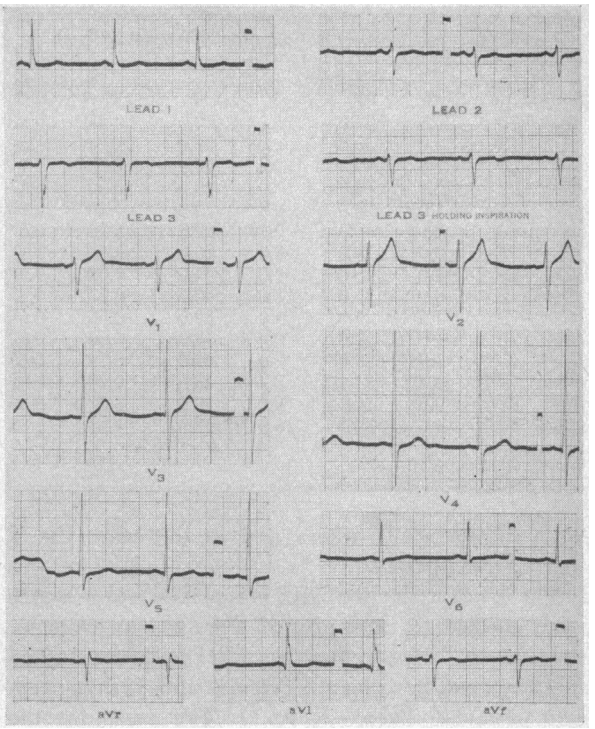

Figure 8.—Electrocardiogram of a patient wtih hypertensive heart disease which shows high voltage of QRS complexes and absence of discordant S-T segments and T waves.

measurement showing the greatest deviation from normal was the cardiac area. The average area for the non-runners was 123 square centimeters as compared with 139 square centimeters for the runners at rest, or an increase of 13 per cent. The predicted transverse diameter of the teleoroentgenogram of the heart of 27 runners at rest was estimated from the body height and weight, using tables of Ungerleider and Clark.[6] Six of the runners had a diameter exceeding the predicted diameter by from 10 to 20 per cent while in one the diameter exceeded the predicted by 35 per cent. The cardiac areas were measured in the same group and predictions were made based on height and weight. Nineteen of the 27 runners had cardiac areas which exceeded the predicted by 10 per cent. In fourteen, the area was from 10 to 30 per cent greater than predicted and in four it was from 31 to 55 per cent above the predicted area.

The electrocardiogram was studied next in patients with strain on the left ventricle to determine if high voltage of the QRS complexes is always associated with discordant S-T segments and T waves. Electrocardiograms of two patients with essential hypertension are shown in Figures 7 and 8. Both of these patients had high voltage (areas greater than average) of the QRS complexes in the leads recorded from the left side of the chest and in both patients there was radiographic evidence of cardiac enlargement. The electrocardiogram of one patient showed the typical left ventricular strain pattern with S-T segments and T waves which were discordant with the main deflection of the QRS complexes (Figure 7). In the other patient the discordant S-T segments and T waves were absent in the electrocardiogram although there was high voltage of the QRS complexes (Figure 8). The areas under the R waves of both of the preceding examples were greater than average; however, in one case there

TABLE 4.—*Measurements of the transverse, broad and long diameters of the heart (in centimeters) and of cardiac area and great vessel diameters (in square centimeters)*

	Transverse		Broad		Long		Area		Great Vessel Diameters	
	Non-runners	Runners	Non-runners	Runners	Non-runners	Runners	Non-runners	Runners	Non-runners	Runners
Mean	121	129	112	117	139	150	123	139	51	54
Min.	104	106	110	105	130	136	112	113	42	46
Max.	139	143	115	132	148	175	136	182	57	64
S.D.±	12	12	0.8	6	6.6	4.8	6.9	14	3.6	4.5
C.V.± %	10	9	0.7	5	4.7	3.2	5.6	10	7.0	8.3

TABLE 5.—*Relationships of areas under the waves of the QRS complexes and T waves in lead V5 in 12 normal subjects*

No.	Heart Rate	Area of Waves* Q	R	S	T	Ratio QRS/T	R/T
1	70	0.0	36.0	16.0	72.0	0.28	0.50
2	75	1.0	50.0	2.0	38.0	1.24	1.32
3	68	1.0	40.0	2.0	36.0	1.03	1.11
4	62	1.0	30.0	0.0	48.0	0.60	0.63
5	75	1.0	20.0	0.0	24.0	0.79	0.83
6	70	1.0	30.0	7.0	92.0	0.24	0.33
7	90	0.0	18.0	8.0	16.0	0.63	1.12
8	65	1.0	45.0	0.0	44.0	1.02	1.02
9	70	0.0	34.0	2.0	40.0	0.77	0.85
10	70	1.0	26.0	4.0	39.0	0.56	0.67
11	70	1.0	30.0	0.0	48.0	0.60	0.62
12	75	1.0	20.0	0.0	24.0	0.79	0.83
Mean	71.7	0.7	31.6	3.4	43.5	0.72	0.82
Min.	62.0	0.0	20.0	0.0	16.0	0.28	0.33
Max.	90.0	1.0	50.0	16.0	92.0	1.24	1.32

*Microvolt seconds.

TABLE 6.—*Relationships of areas under the waves of the QRS complexes and T waves in lead V5 of 12 runners*

No.	Heart Rate	Area of Waves* Q	R	S	T	Ratio QRS/T	R/T
1	60	3.0	80.0	0.0	68.0	1.13	1.18
2	42	0.0	92.0	7.0	120.0	0.71	0.77
3	50	2.0	60.0	24.0	60.0	0.57	1.00
4	60	0.0	50.0	10.0	60.0	0.65	0.83
5	60	2.0	60.0	10.0	100.0	0.48	0.66
6	60	1.0	90.0	5.0	228.0	0.37	0.39
7	58	2.0	62.0	0.0	104.0	0.58	0.59
8	60	0.0	70.0	3.0	112.0	0.58	0.63
9	55	1.0	70.0	15.0	72.0	0.75	0.97
10	45	1.0	70.0	4.0	92.0	0.71	0.76
11	65	1.0	60.0	12.0	56.0	0.84	1.07
12	45	1.0	56.0	7.0	76.0	0.64	0.74
Mean	55	1.4	68.3	8.0	95.7	0.67	0.79
Min.	42	0.0	50.0	0.0	56.0	0.37	0.39
Max.	65	3.0	92.0	24.0	228.0	1.13	1.18

*Microvolt seconds.

were discordant T waves and in the other the T waves were upright.

The areas under the QRS complexes and T waves were studied to determine if there is any constant relationship between these waves. They were studied in 12 normal persons, 12 runners and 12 hypertensive patients (Tables 5, 6, 7). The areas for the Q, R, S and T waves were determined separately in lead V5. The differences in the areas between the positive and negative waves of the QRS complexes were calculated. The QRS/T and R/T ratios were determined. The average for 12 normal subjects is shown in Table 5. Q waves were present generally but were small, averaging 0.7 microvolt second. The average R wave area was 31.6 microvolt seconds while the average T wave area was 43.5 microvolt seconds. The QRS/T ratio averaged 0.72; the R/T ratio averaged 0.82, which means that the areas under the R waves were nearly equal to the areas under the T waves. In the runners, the Q waves were slightly larger, averaging 1.4 microvolt seconds (Table 6). The average area under the R waves was greater, averaging 68.3 microvolt seconds, while the average area under the T waves was 95.7 microvolt seconds. The QRS/T ratio averaged 0.67. The R/T ratio averaged 0.79, being similar to that found in the normal persons. Among the hypertensive subjects, the Q waves were slightly increased over normal, being 1.1 microvolt seconds (Table 7). The areas under the R waves were less than they were in the runners, averaging 64.0; however, the T waves were distinctly lower, the average being a negative value of 55.0 microvolt seconds. It was not possible to calculate the QRS/T or R/T ratios in eight of the 12 hypertensive patients, as the T waves were discordant with the R waves. When the R/T ratio could be calculated, it averaged 8.7, which indicates that the T waves, when upright, are low compared to the amplitude of the R waves. The

TABLE 7.—*Relationships of areas under the waves of the QRS complexes and T waves in lead V5 in 12 patients with hypertensive heart disease*

No.	Heart Rate	Area of Waves* Q	R	S	T	Ratio QRS/T	R/T
1	60	0.0	70.0	20.0	36.0	1.39	1.89
2	60	0.0	50.0	24.0	—44.0	.....	.....
3	70	1.0	48.0	2.0	—24.0	.....	.....
4	80	0.0	75.0	0.0	72.0	1.04	1.04
5	85	2.0	50.0	2.0	—28.0	.....	.....
6	60	2.0	60.0	0.0	2.0	30.00	29.00
7	60	1.0	75.0	0.0	—14.0	.....	.....
8	80	2.0	50.0	0.0	—12.0	.....	.....
9	60	2.0	75.0	14.0	—32.0	.....	.....
10	58	1.0	68.0	0.0	—44.0	.....	.....
11	100	2.0	85.0	0.0	—10.0	.....	.....
12	60	1.0	60.0	35.0	32.0	1.06	1.87
Mean	69.4	1.1	64.0	8.0	—55.0	8.12	8.70
Min.	58.0	0.0	48.0	0.0	—44.0	1.04	1.04
Max.	100.0	2.0	85.0	35.0	36.0	29.00	30.00

*Microvolt seconds.

QRS/T ratio averaged 8.12. In certain disease states there is high voltage of the QRS complexes without discordant S-T segments and T waves. Of interest is hypertensive patient No. 4 (Table 7) who had high voltage of the QRS complex in lead V5 and who had a normal R/T ratio. The area under the R waves was 75.0 microvolt seconds and under the T wave 72.0 microvolt seconds. This gives an R/T ratio of 1.04. The electrocardiogram in this case was similar to that of the runners and was also similar to that of a patient with rheumatic aortic stenosis and insufficiency who also had high voltage of the QRS complexes with a normal R/T ratio (Figure 9). In ten adult patients with coarctation of the aorta there was increased voltage of the R waves in the left precordial leads; however, three of these patients had normal R/T ratios.

These findings may be summarized by stating that the normal subjects and runners had R/T ratios that approached 1.0, which means that the areas under the R waves and T waves are nearly equal and these waves are of similar polarity; however,

TABLE 8.—*The relationship between the type of electrocardiogram and symptoms and signs of heart disease in 30 hypertensive patients with high voltage of QRS complexes. The areas under the R waves in the two groups are similar.*

Symptoms or Signs	Normal R/T Ratios* (Concordant S-T segments and T waves)	Abnormal R/T Ratio* (Discordant S-T segments and T waves)
Dyspnea	10	60
Angina Pectoris	3	72
Cardiac Enlargement	20	60
Congestive Heart Failure	0	40
Average blood pressure	185/110	210/124

*Per cent of 30 patients.

the runners had R waves and T waves in the left precordial leads, with areas that are essentially twice as large as in the normal subjects. Hypertensive patients usually had S-T segments and T waves that are discordant with the R waves or had an abnormal R/T ratio.

The relationship between the electrocardiogram and symptoms and signs of heart disease was studied in 30 hypertensive patients all of whom had high voltage of the QRS complexes (Table 8). These patients were divided into two groups, those with normal R/T ratios and those with abnormal R and T relationships. The areas under the R waves in lead V5 were similar, averaging 68.0 and 70.0 microvolt seconds, respectively. The average blood pressures for the two groups were 185/110 in the former and 210/124 in the latter. It can be seen (Table 8) that dyspnea, angina pectoris, cardiac enlargement and congestive heart failure occurred more frequently in patients with discordant T waves than in those who had concordant T waves. Thus it is evident that patients with abnormal R/T ratios have a greater incidence of signs and symptoms of hypertensive heart disease than do the patients with normal R/T ratios.

DISCUSSION

The electrocardiogram of the marathon runner at rest is characterized by a slow cardiac rate, usually below 60 beats per minute, high voltage of the R waves in leads 2, V4 and aVf and deep S waves in the precordial leads recorded from the right side of the chest. The sum of the amplitude of the R

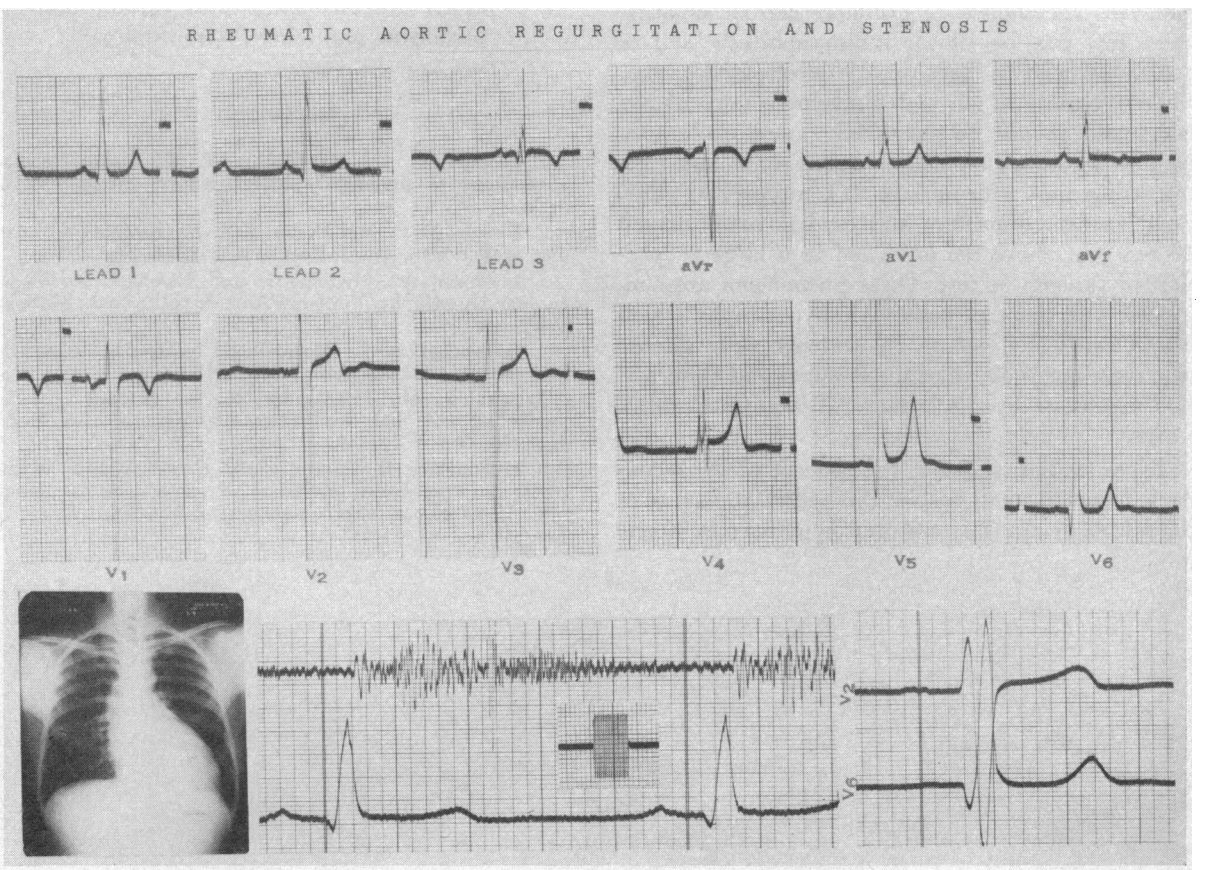

Figure 9.—Electrocardiogram and cardiac silhouette of a patient with rheumatic aortic regurgitation and stenosis. High voltage of the QRS complexes and increase in amplitude of the T waves is present. Teleoroentgenogram shows left ventricular enlargement. Lead V2 and V6 taken simultaneously with a paper speed of 75 millimeters per second show a delayed onset of the intrinsicoid deflection in lead V6 suggesting left ventricular enlargement. Stethogram shows typical systolic and diastolic murmurs over the aortic area.

waves in lead V5 added to the depth of the S waves in lead V1 exceeded the upper limit of normal in a significant percentage of the runners. The T waves were tall and a normal R/T ratio was present in all runners. R-R prime waves occurred occasionally in lead V1. All of these changes are considered characteristic of left, and to a lesser degree, of right ventricular enlargement. The presence of cardiac enlargement in runners at rest was confirmed by an increase in the cardiac area. Immediately after they had been running, the voltage of the QRS complexes and T waves increased, while the transverse cardiac silhouette decreased in all its diameters, the heart apparently being in a state of increased muscular tone. The amount of air space seen between the heart and the chest wall was not altered detectably after running. Therefore the increased voltages after running appear to be due to an increased potential from the myocardium itself rather than to a positional change of the heart, or to a more proximal position of the electrodes on the chest to the surface of the heart.

It is important to recognize that athletic exercise that calls for endurance produces cardiac enlargement that can be shown radiographically and is associated with a characteristic electrocardiogram. This is typical of physiologic hypertrophy of the heart and should be suspected among endurance athletes who have typical roentgenograms and electrocardiograms with normal R/T ratios. The transverse cardiac diameters and cardiac areas exceed 10 per cent above the predicted in a large proportion of runners at rest. These phenomena are contrasted with those of pathologic hypertrophy which characteristically occurs in patients with disease who have high voltage of the R waves and abnormal R/T ratios or by discordant S-T segments and T waves.

These studies challenge the concept of the secondary T wave in patients with increased pressures in the left ventricle (*i.e.*, essential hypertension, aortic valvular disease and other cardiac diseases). In early cases of hypertension there seems to be strain on the left ventricle with increased voltage of the QRS complexes without alteration of the T waves. This produces an electrocardiogram similar to that produced by physiologic hypertrophy of the heart such as occurs in marathon runners. Among these patients the complications and signs and symptoms of hypertensive heart diseases are few. The incidence of complications, signs and symptoms is much higher among those with discordant S-T segments and T waves. These phenomena suggest that the discordant T waves in hypertension are primary and are due, most likely, to myocardial disease. Anoxia of the endocardium, especially during systole, may be responsible for these changes, especially in patients with hypertension. Schoenmakers[4] observed that in hypertension there is an increase in the cross-sectional area of myocardial cells with a decrease in the number of capillaries per unit of tissue. This would result in chronic anoxia of the myocardium. These or other factors could be responsible for primary discordant S-T segments and T waves which are encountered in certain patients with hypertension and other disease states.

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